1100-41 An Angiographic Risk Score Integrating Both Epicardial and Tissue Level Perfusion Before and After Facilitated Percutaneous Coronary Intervention in Acute MI

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Background: Both epicardial and tissue level perfusion have been related to clinical outcomes in the setting of acute myocardial infarction (AMI), and the performance of adjunctive/rescue percutaneous intervention may alter clinical outcomes after thrombolytic administration. ***Objectives & Methods:*** The goal of this study was to develop a simple, broadly applicable method that integrates epicardial and tissue level perfusion both before and after PCI to arrive at a single angiographic risk score (ARS) in patients undergoing PCI after thrombolysis. The angiographic risk score is the arithmetic sum of the TIMI Flow Grade (0-3) added to the TIMI Myocardial Perfusion Grade (0-3) before and after PCI (total possible score of 0-12). This risk score was evaluated in patients from the LIMIT AMI trial of tPA monotherapy vs tPA plus rhuMAb CD18. Infarct size was assessed using 120-216 hr post-AMI SPECT Technetium-99m Sestamibi data. ***Results:*** Those patients with an angiographic risk score in the lowest group (0-6) had a risk of 30 day death or MI of 9.3% (5/54), whereas those with an ARS of 7-12 had a risk of 1.3% (1/79)(p=0.04). There were no deaths or recurrent MIs among patients with a risk score greater than 10. Likewise, larger SPECT infarct sizes were observed among patients with an ARS of 0-6 (22.6% ± 20.4%, n=53) compared to those patients with an ARS of 7-12 (12.3% ± 14.3%, n=71, p=0.001). In a second analysis, data from patients who did not undergo PCI was incorporated by using the final TIMI Flow Grade and the final TIMI Myocardial Perfusion Grade on diagnostic arteriography instead of the post PCI values, and similar results were seen: the risk of 30 day death or MI was 11.7% (11/94) for ARS of 0-6, whereas it was 4.2% (6/143) for ARS of 7-12. SPECT infarct sizes were larger for ARS of 0-6 (21.0 + 19.0, n=84) vs ARS of 7-12 (11.8 + 15.2, n=127, p=0.0001). ***Conclusions:*** The angiographic risk score integrates indexes of epicardial and tissue level perfusion before and after PCI or at the end of diagnostic cardiac catheterization to arrive at a single risk estimate that is associated with infarct size and 30 day death or MI. Failure to achieve an ARS of > 6 is associated with a doubling of infarct size.

1100-42 Myocardial Perfusion in Patients With Non-ST-Segment Elevation Acute Coronary Syndrome Assessed With Venous Contrast Echocardiography

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Non ST segment elevation acute coronary syndrome (NSTEACS) is a dynamic condition and the underlying pathophysiology is currently thought to comprise not only atherosclerotic plaque rupture and superimposed thrombosis of an epicardial coronary artery but also microvascular obstruction to flow due to vasoconstriction, microembolisation and adherence of activated blood cells to microvascular endothelium. We therefore sought to compare venous myocardial contrast echocardiography (VMCE), a new bedside technique allowing for exclusive imaging myocardial vasculature, to Sestamibi SPECT (SI), angiographic and biochemical findings in patients with NSTEACS.

60 patients (women, n=15, age 65±11y) with typical anginal chest pain at rest or during minimal physical activity plus either transient EKG changes (without ST segment elevation) or elevated Troponin T (> 0.03 µg/ml) on presentation were included in the study. All patients underwent VMCE (3ml Optison in 27ml NaCl, 200ml/h, intermittent harmonic imaging every 5th endsystole, off-line digital image processing), SI and coronary angiography within 1 hour after presentation. 52/60 patients (87%) had myocardial contrast defects on VMCE. Concordance with respect to presence of myocardial defects on SI was 88% (k=0.74). 26/60 patients (43%) had TIMI flow <3; all of these patients had a contrast defect on VMCE and 17/26 patients (65%) had elevated Troponin I. 34/60 patients (57%) had TIMI flow 3; 26/34 patients (76%) had a contrast defect on VMCE and 14/34 patients (41%) had elevated Troponin I.

We conclude that VMCE is a promising method for evaluation of myocardial perfusion in patients with NSTEACS. It is reasonable to further investigate its role for diagnostic workup and follow up of patients with NSTEACS.

1100-43 Significant Myocardial Salvage in Patients With Non-ST Elevation Myocardial Infarction Is Common: Results Using Serial Myocardial Perfusion Imaging

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Background: Technetium-99m sestamibi can delineate both myocardium at risk and final infarct size in patient (pts) with myocardial infarction (MI). Serial myocardial perfusion imaging (MPI) in pts with non-ST elevation MI has not been described previously. **Methods:** Pts with non-ST elevation MI who had MPI at the time of Emergency Department (ED) presentation were included. Percent defect size was quantitated using a 50% threshold derived from a phantom (r=0.99) using multiple short axis slices. Myocardial risk area was defined as the initial defect size; infarct area as the defect size on repeat MPI; and myocardial salvage as the difference between the two. Ejection fraction (EF) was calculated using a previously validated computer algorithm (QGS). **Results:** There were 69 pts who had acute ED MPI and MI who underwent repeat MPI a median 5 days later. Revascularization was performed in 46 pts (67%) (only 2 within 12 hours of presentation). Variation in both mean peak CK (704±1223 U/L, median 377 U/L, range 91 to 9319 U/L) and risk area (19±10%, median 19%, range 2-46%) was high. Mean final infarct size, 11±+9% (median 9%, range 0-35%), was significantly smaller (p<0.001), and was only 57% of the initial risk area. Significant salvage (initial risk area-final infarct size >25%) occurred in most pts (67%), with 54% of pts having > 50% salvage. Pts with significant salvage had an increase in EF (48 to 55%, 22% increase, p<0.01), while those without significant salvage EF decreased (49 to 48%, 2% decrease, p=NS). **Conclusions:** The ischemic risk area in pts with non-ST elevation MI can be large. Significant myocardial salvage is common, is not limited to those that have early revascularization, and is associated with improved EF.

1100-44 Old Age and Myocardial Infarct Size in the Reperfusion Era

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It is well established that cardiac death and complications are more common in elderly patients who suffer myocardial infarction. Although the mechanism for this increased morbidity and morality is unknown, one potential explanation, extrapolated from some experimental models of myocardial ischemia/reperfusion, is that an age-associated increase in infarct size may play a role. Data on infract size in old versus young patients are lacking. We examined whether CK-MB determined myocardial infarct size differed in elderly (65 years or older) versus younger (< 65 years old) patients who did not have coronary reperfusion (from the prethrombolytic era MILIS study) and from the more recent thrombolytic TIMI 4 trial. Infarct size data in MILIS (MB infarct size index = area under MB-CK curve adjusted for body surface area) was collected in 639 patients < 65 and 213 elderly patients. Mean MB infarct size index was 15.59 ± 0.59 Units for those < 65 and 11.89 ± 0.83 Units for elderly patients (p = 0.0003). Infarct size data in TIMI 4 (MB-CK = average MB-CK over first 14 hours) was collected in 260 patients < 65 and 140 elderly patients. Mean MB-CK infarct size was 123.2 ± 8.3 International Units per liter (IU/L) in patients < 65 and 119.5 ± 10.4 IU/L for elderly patients (p = 0.78). The smaller infarcts in older MILIS patients were not due to lower presenting heart rates which were 79.8 beats/min (mean) in elderly patients and 80.4 for younger patients (p = NS); mean heart rates in TIMI 4 were 73.4 in elderly patient and 71.1 in younger patients (p = NS). These data suggest that in the absence of reperfusion, infract size is smaller (rather than larger) in older patients. Reperfusion changes this relationship resulting in equally sized infarcts in old versus young, suggesting that in the reperfusion era, infarct size is not larger in elderly patients and probably does not contribute to their worse outcome.

1100-45 Rarity of Circumflex Culprits in ST-Elevation MI is Due to Relative ECG Silence

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Background: Angiographic studies of ST-elevation myocardial infarction (MI) consistently document that the left circumflex coronary artery (CFX) is uncommonly the culprit vessel compared to the left anterior descending (LAD) and the right coronary (RCA). Since there is no a priori reason to expect a differential propensity of the three coronary vessels to acute plaque instability, we hypothesized that this disparity of CFX culprits may result from ECG insensitivity in detection of lateral wall transmural ischemia. If this hypothesis is correct, the distribution of culprit vessels in non ST-elevation acute coronary syndromes(ACS) would be expected to be relatively even. **Methods:** We retrospectively analyzed angiograms from our cath lab database to identify the culprit vessel in 166 pts with ST-elevation MI and in 134 others with non-ST elevation ACS. **Results:** In pts with ST-elevation MI, the CFX was uncommonly the culprit vessel (12%), compared to LAD (43%) or RCA (45%) involvement (CFX vs LAD or RCA, p<0.001). However, in pts with non-ST elevation ACS, the distribution of culprit vessels was more even and in fact the CFX was most frequently the culprit vessel (40%), whereas the LAD was responsible in 28% of pts and the RCA in 32% of cases (CFX vs LAD or RCA, p=NS). Compared to pts with ST-elevation, in those with non-ST elevation ACS the CFX was more commonly the culprit vessel (40 vs 12%, p<0.001). The frequency of LAD and RCA culprits was similar between the two groups. **Conclusions:** These findings document a striking disparity in the prevalence of CFX culprits in pts with ST-elevation MI compared to non-ST elevation ACS. The rarity of CFX culprits in ST-elevation MI but even distribution in other ACS, suggests that the CFX is just as likely to develop plaque rupture but that lateral wall transmural ischemia is often missed by ECG.

1100-46 Does Prior Angina Predict Outcomes Following Acute Myocardial Infarction? Testing the Relevance of Ischemic Pre-Conditioning in the Era of Primary Angioplasty

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Background: In animal studies, brief periods of myocardial ischemia reduce the size of the infarct resulting from a subsequent total occlusion. It is postulated that angina preceding acute myocardial infarction (AMI) may provide similar benefit in humans. However, clinical studies on the prognostic impact of preinfarction angina have shown mixed results.

Methods: We tested the effect of prior angina on in-hospital outcomes [ischemic target vessel revascularization (TVR), reinfarction, death, & MACE (combined end-point)] in the pooled database of 2558 patients enrolled in Primary Angioplasty in Myocardial Infarction (PAMI)-2 & Stent PAMI trials. To minimize confounding from silent ischemia & atherosclerotic burden, we excluded 878 (34%) patients with a history of diabetes, prior myocardial infarction, prior percutaneous coronary intervention or coronary artery bypass surgery from this analysis. In the remaining 1680 patients, we compared angiographic & clinical outcomes between patients with (n=230, 17%) & those without prior angina (n=1450, 83%).

Results: Prior angina patients had a higher incidence of hypertension (50 vs 40%), dyslipidemia (45 vs 33%), peripheral vascular disease (5.7 vs 3.0%), & previous aspirin use (24 vs 9%, p<0.05 for all). The 2 groups had otherwise similar baseline clinical & angiographic features. Stent use (29 vs 29%), final TIMI-3 flow (91 vs 92%), mean residual stenosis (22 vs 21%), & cath lab complications (33 vs 29%) were comparable between groups. Patients with prior angina had similar incidence of in-hospital reinfarction (1.3 vs